

MUST DO PHARM IN FOUNDATION PHASE

Watch the following topics from the recommended sources (Sketchy, Osmosis, Dirty Medicine on YouTube):

1. Cardiac Pharmacology:

- Antiarrhythmics and Digoxin – Sketchy

2. Lipid-Lowering Agents:

- Lipid-lowering drugs – Dirty Medicine (optional, if needed for clarification)

3. Endocrine Pharmacology:

- Insulin – Sketchy
- Oral hypoglycemics – Osmosis (recommended if you're unsure about the mechanisms of action)

4. Neuro/Psych Pharmacology:

- Antipsychotics, Anticonvulsants, and Anti-Parkinsonism drugs – Osmosis (focus on MOA and side effects), MORPHINE
- SSRIs and Atypical Antidepressants – Osmosis (focus on MOA and side effects), SNRI, TCA, Lithium.

5. Anesthetics:

- General Anesthetics – Osmosis (MOA and side effects)

6. Renal Pharmacology:

- Loop Diuretics, Thiazides, and Spironolactone – Sketchy

7. Immunology/Allergy:

- Antihistamines – Osmosis

⑧ → Vancomycin, Metronidazole, Fluoroquinolone, Tetracycline (Anti-TB) (TMP-SMX) → Osmosis

→ ONDANSETRON → Pixorize.



Digoxin, milrinone, nesiritide

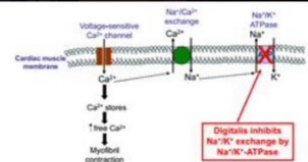
- DJ Foxglove: Digoxin** – Derived from the foxglove plant
Knocked over banana vending machine: inhibition Na^+/K^+ ATPase
- Obstruction salty sodium peanuts: increased intracellular sodium as a result of Na^+/K^+ ATPase inhibition
- Salty peanuts sneaking in the calci-yum ice cream: increased intracellular sodium promotes calcium influx at the $\text{Na}^+/\text{Ca}^{2+}$ exchanger
- Flexed arm: increased cardiac contractility due to the positive inotropic effects
- Deflated heart balloon: symptomatic treatment of chronic systolic heart failure. Only used for symptomatic relief, does not decrease mortality
- Las Vegas: direct stimulation of the vagus nerve allows for treatment of Atrial arrhythmias
- Rhythm-inducing record: antiarrhythmics
Patients presenting will have HF and A.Fib, labs show elevated in K^+
- Adverse effects**
- Pile of bananas: Hyperkalemia with acute digoxin toxicity
- Various dances on the heart shaped dance floor: digoxin may induce various arrhythmias
- TaSTy scoop: chronic digoxin use may cause scooped concave ST segments on EKG
- SA music note: side effect of bradycardia due to parasympathetic activity of SA node
- Dangling heart watch: side effect of bradycardia
- Av MUSIC NOTE: SIDE EFFECT OF HEART BLOCK DUE TO DIGOXIN TOXICITY
- Remain Unblocked: Digoxin is contraindicated in SA node heart block, or in use with caution in Beta Blockers**
- GI side effects include nausea, vomiting, and abdominal pain
- Yellow spotlight: side effect of Xanthopsia (objects appear yellow)
- Kid stuffed inside banana depleted vending machine: Hypokalemia will exacerbate digoxin toxicity
- Loop diuretics can cause hypokalemia, along with diarrhea or vomiting may also occur
- Kidney jukebox with long tapering flag on cracked kidney: Renal insufficiency can make digoxin toxicity worse and will precipitate digoxin rise, the long flag indicates the long $\frac{1}{2}$ life of digoxin, an increasing susceptibility to toxicity
- Records in kidney jukebox: many arrhythmias inhibit renal clearance of digoxin, increasing susceptibility to toxicity
- Fabulous: digoxin immune fab is used to reverse toxicity
- One in a million: Milrinone**
- Don't phoster disinterest: milrinone inhibits phosphodiesterase
- CAMPain: milrinone decreases CAMP breakdown
- Flexing arms: milrinone increases cardiac contractility
- Dilated red donkey ears: milrinone causes arteriolar dilation in HF, but watch for hypertension
- Turn the tide: nesiritide**
- BuMP: BNP analog that increases cGMP
- Dilated red ears and blue legs: nesiritide causes arteriolar and venous dilation, reducing afterload and preload
- Salty peanut stream: nesiritide causes natriuresis

Digoxin



Class V: Rate control

- DJ Foxglove: Digoxin has antiarrhythmic properties**
- Vegas: digoxin exerts direct parasympathetic effects via direct stimulation of the vagus nerve → AV nodal inhibition
- Irregularly irregular signal: digoxin is useful in atrial fibrillation and flutter, not first line
- Metronome: digoxin prevents rapid ventricular response in atrial fibrillation and flutter (rate control)
- Magnets: magnesium** is useful for the treatment of certain arrhythmias (torsades)
- Torn twisted torsades streamers: magnesium treats torsades de pointes
- Banana dancer pointing up: hyperkalemia can induce arrhythmia
- Peaked streamer: hyperkalemia can cause peaked T waves (with shortened QT intervals) on ECG
- Banana dancer pointing down: hypokalemia can induce arrhythmias, severe muscle weakness, and glucose abnormalities
- Streamer with extra bump: hypokalemia can induce U waves at the end of the T wave on EKG
- Swing dancing: adenosine** (a purine nucleoside with antiarrhythmic properties)
- Purine shaped gate: adenosine is a purine nucleoside
- A1 swing: adenosine activated inhibitory A1 receptors on the myocardium and at the SA and AV nodes
- Banana flying out of the cup: activation of A1 receptors increases outward K^+ current (hyperpolarizes, suppressed, Ca^{2+} dependent AP)
- Falling calci-YUM ice cream: activation of A1 receptors suppress inward Ca^{2+} current
- Note shaped dance floor: Adenosine inhibits AV nodes (decreased AV conduction, prolonged AV refractory period)
- Hat blocking heart: Adenosine causes transient high grade heart block (direct av node inhibition for about 10s)
- Disconnected bottom of heart: adenosine decreases atrioventricular conduction
- Illuminated top of heart: adenosine is a first line agent for acute treatment of supraventricular arrhythmias (PVST)
- Dilated coronary crown: adenosine causes coronary dilation (mediated by A2 receptors)
- Adverse Effects**
- Flushed dancer: adenosine can cause cutaneous flushing
- Dancer clutching chest: adenosine can cause shortness of breath, chest pain, and impending sense of doom
- Fainting dancer: adenosine can cause fainting, headache, and hypotension
- Energy drink blocking A1 gate: the actions of adenosine are inhibited by caffeine and theophylline (methylxanthines)



Adenosine

Cardiovascular and Kidney

Class 1 A.

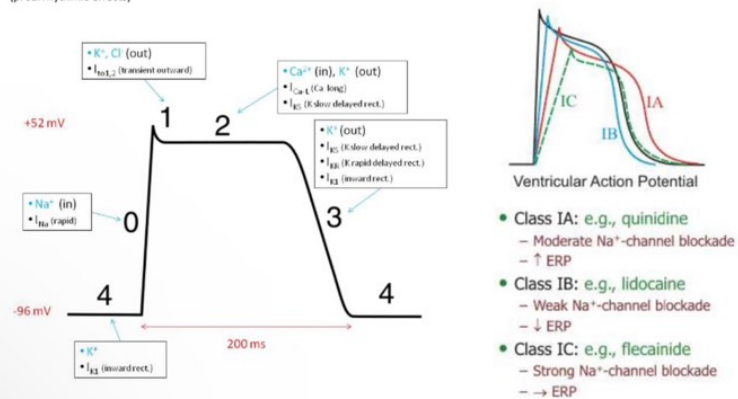


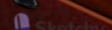
Antiarrhythmic Class I A-C (rhythm control): No (Class I sodium) Bad Boy (Beta Blockers) Keeps (Potassium) Clean (calcium)

- Soloist: class I antiarrhythmics
- Microphone stand Phase 0: of the AP upstroke dictated by Na^+
- Wire off the microphone is Phase 2: plateau dictated by Ca^{2+}
- Phase 3 downstroke: repolarization dictated by K^+
- Soloist holding peanut jar: Class I antiarrhythmic block sodium channels
- Soloist tipping mic stand: class I antiarrhythmics decrease the slope of phase 0 upstroke (slows conduction of the cardiac AP) AP will almost look tipped over with a decreased slope
- Inactivating spoon in open peanut butter jar: Class I antiarrhythmics bind to open or inactivated Na^+ channels
- Heart tipping mic stand: "use dependence" – class I antiarrhythmics have a greater effect on rapidly depolarizing tissues (increased heart rate causes slower phase 0 upstroke)
- Potassium banana curtain: K^+ current present during Phase 2 (plateau) and phase 3 (repolarization) of the cardiac action potential
- Illuminated atria, ventricles, and His-perkinje system: class I antiarrhythmics affect the Na^+ dependent cardiac action potential (no action at the SA and AV nodes)
- Wide QRS shadow: class I antiarrhythmics widen the QRS complex on the ECG (decreased AP conduction velocity) this will happen when the HR increases because that will increase the effect of the drug
- Class IA antiarrhythmics: quinidine, procainamide, disopyramide "Double, Quarter, Pounder"**
- Dining prom queen: quinidine (class IA antiarrhythmic)**
- Prom King: procainamide (class IA antiarrhythmic)**
- "Disappears!" disopyramide (class IA antiarrhythmic)**
- Binding strength: IC-IA-IB
- Prom queen lightly holding peanut butter jar: class IA antiarrhythmics have an intermediate binding affinity to the Na^+ channel (intermediate use dependence, moderate slowing of phase 0 upstroke) **increased AP duration**
- Pushing away the curtain: class IA antiarrhythmics also block K^+ channels prolonging phase 2 and 3 of the cardiac action potential → prolonged refractory period
- Illuminated top and bottom of IA heart: class IA antiarrhythmics treat supraventricular and ventricular arrhythmias
- White wolf pack: class IA antiarrhythmics treat WPW syndrome (a type of SVT involving extra signals in a accessory pathway)
- Tin cans: quinidine toxicity can cause cinchonism (syndrome of tinnitus, headache, dizziness)
- Broken plates: quinidine can cause thrombocytopenia
- Prom king's lupus wolf: procainamide can cause a lupus-like syndrome raising an ANA titer
- Darts in failing heart balloon: disopyramide can exacerbate heart failure (negative inotropy)
- Twisted torsades streamer: Class IA antiarrhythmics can cause Q-T interval prolongation (Precipitates torsades)
- Whenever the potassium current is prolonged and thrown aside it can cause torsades de pointes (prolonged QT interval)
- Class IB antiarrhythmics "Lettuce, Tomato, Mayo"**
- Lied: lidocaine (class IB antiarrhythmic)**
- Friendly toward: Phenytoin (an anti-epileptic) that shows some type IB properties**
- Mexican flag: mexilitine (class IB antiarrhythmic)**
- Dropped peanut butter jar: Class IB antiarrhythmic have a low binding affinity for the Na^+ channel (low use dependence, modest slowing of the phase 0 upstroke) **decreased AP duration**
- Pulling the curtain: class IB antiarrhythmics shorten phase 2 and 3 of the cardiac action potential → shortened refractory period so no chance for torsades de pointes
- Illuminated and cracked bottom of heart: Class IB antiarrhythmics treat ventricular arrhythmias (especially in ischemic tissues) in sodium channels spending more time in the open and resting state because of the longer action potential
- Broken illuminated IB heart: has a greater tendency to work with ischemic heart because of the reduced resting membrane potential delays sodium channel transition from inactive back into resting state resulting in increased drug binding
- "DEAD": class IB antiarrhythmics treat ischemia induced ventricular arrhythmias one of the most common causes of death in the acute period following an MI**
- Brain trucker hat: class IB antiarrhythmics cause neurological problems (parasthesias, tremor, convulsions)

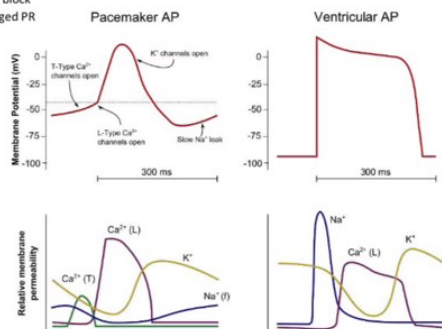


- Class IC antiarrhythmics Heart floor: propafenone, flecainide "Fries Please"**
- Flakes: flecainide (class IC antiarrhythmic)**
- Purple phone: propafenone (class IC antiarrhythmic)**
- Tightly held peanut butter jar: Class IC have a strong binding affinity for the Na^+ channel (strong use dependence, drastic slowing of the phase 0 upstroke) **dramatic effect on QRS duration, prolongs ERP in AV node, no change in AP Duration**
- Untouched potassium curtain: class IC antiarrhythmics do not affect the cardiac action potential duration
- Illuminated top and bottom of heart: class IC antiarrhythmics treat supraventricular (A.Fib) and ventricular arrhythmias
- Irregularly irregular signal: Class IC antiarrhythmics treat atrial fibrillation (and flutter)
- Converting the signal: class IC antiarrhythmics can restore and maintain normal sinus rhythm in A. Fib and Flutter
- "Healthy Hearts Only": class IC antiarrhythmics are contraindicated in patients with history of structural or ischemic heart disease (proarrhythmic effects)**





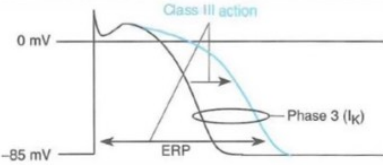
1. Due: class II antiarrhythmics
2. Muted beta bugle: beta blockers (class II antiarrhythmics)
3. Notes: beta blockers treat arrhythmias by blocking sympathetic input to SA and AV nodes
4. Torn Band Camp: beta blockers decrease cAMP
5. Crushed calci-um ice cream cartons: decreased cAMP leads to closure of membrane calcium channels, preventing the upstroke of AV nodal action potential
6. Phase 4: pacemaker current dictated by Na^+ (funny current) and other ion. Depolarization is from Ca^{++}
7. Phase 0: upstroke dictated by Ca^{2+}
8. Phase 3 repolarization dictated by K^+
9. Sliding up the keys: beta blockers prolong phase 4 of the nodal action potential decreased pacemaker activity, prolonged conduction time and refractory period
10. Disconnected bottom of light: beta blockers decrease atrioventricular conduction
11. Heart light lit up top: beta blockers treat supraventricular arrhythmias (Afib and RVR)
12. IVY: IV beta blockers (esmolol) can be used for acute supraventricular arrhythmias
13. Hat shielding heart: beta blockers can cause heart block
14. Public relations: heart block manifests as a prolonged PR interval on EKG
15. Irregularly irregular static signal: beta blockers are useful in atrial fibrillation (and flutter)
16. Metronome: beta blockers prevent rapid ventricular response in atrial fibrillation and flutter "rate control" but does not fix the atrial fibrillation



Class 2



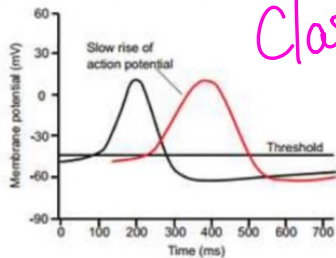
- Phase 2: plateau dictated by Ca^{2+}
- Phase 3: repolarization dictated by K^{+}
- Potassium banana theme curtain: K^{+} current present during Phase 2 (plateau) and Phase 3 (repolarization) of the cardiac action potential
- Singer pushing away the curtain: class III antiarrhythmics block K^{+} channels prolonging phase 2 and 3 of the cardiac action potential → prolonged refractory period
- "uno, dos, tres, quattro": Amiodarone shares properties of class I, II, III, and IV antiarrhythmics.
- "till I die": -tilide suffix shared by dofetilide and ibutilide (class III antiarrhythmics)
- Soda: sotalol
- Muted bugle and soda bottles: sotalol is also a beta blocker (lol suffix)
- Heart illuminated on top and bottom: class III antiarrhythmics treat both supraventricular arrhythmias and also ventricular arrhythmias
- Irregularly irregular signal: class III antiarrhythmics treat atrial fibrillation and flutter
- Converting the signal: class III antiarrhythmics can restore and maintain normal sinus rhythm in atrial fibrillation and flutter
- Adverse effects**
- Skull brains: amiodarone has many neurologic side effects (tremor, ataxia, peripheral neuropathy, sleep disturbances)
- Gay sunglasses: amiodarone can cause grey corneal deposits
- Bag and small bottles: amiodarone can cause hyper or hypothyroidism, always evaluate thyroid prior to use and monitor during
- Fibrotic lung embroidery: amiodarone can cause pulmonary fibrosis
- Tight button: amiodarone induced lung fibrosis causes restrictive lung diseases
- Trampled failing heart balloon: amiodarone can induce heart failure
- Liver spot: amiodarone can cause hypersensitivity hepatitis (always monitor LFT's)
- Grey blue outfits: amiodarone can cause gray blue skin discoloration
- Flash photo: amiodarone can cause photo dermatitis
- Broken chrome bumper: amiodarone inhibits the cytochrome P450 inhibition
- Twisted streamer: sotalol, dofetilide, and ibutilide can induce dose related torsades (although all type III antiarrhythmics can widen the QT interval)



Class 3



- 4 singers quartet: Class IV (rate control at SA and AV nodes) Non-dihydropyridine calcium channel blockers
- L shaped nozzles on wall: Block L type calcium channels in the heart
- Nondairy: non-dihydropyridine calcium channel blockers (class IV antiarrhythmics)
- Delicious dark chocolate: Diltiazem (non-dihydropyridine CCB)
- Very Vanilla: verapamil (non-dihydropyridine CCB)
- Notes on music sheet: Exert a greater effect on tissues that fire more frequently that use a calcium current, non-dihydropyridines treat arrhythmias by blocking Ca^{2+} current in the SA and AV nodes
- Keys leading up the piano as the gradual phase 4: pacemaker dictated by Na^{+} and other ions
- Phase 0: upstroke dictated by Ca^{2+}
- Phase 3: repolarization dictated by K^{+}
- Sliding up the keys: non-dihydropyridine CCB's prolong phase 4 of the nodal action potential $\rightarrow$ decreased pacemaker activity, prolonged conduction time and refractory period
- Disconnected bottom: non-dihydropyridine CCB's decrease atrioventricular conduction
- Illuminated top: non-dihydropyridine CCB's treat supraventricular arrhythmias (A.Fib with RVR)
- Public relations: non-dihydropyridine CCB's will prolong the PR interval
- Hat shielding head: Non-dihydropyridine CCB's can cause heart block, be careful when combining with other drugs that cause AV nodal blocking like digoxin
- Irregularly irregular signal: non-dihydropyridine CCB's are useful in atrial fibrillation and flutter
- Metronome: non-dihydropyridine prevents rapid ventricular response in A.Fib and Flutter



Class 4

Statin & Others.

Drug	Main Target
HMG CoA Reductase Inhibitors	↓ LDL
Bile Acid Resins	↓ LDL
Ezetimibe	↓ LDL
Fibrates	↓ Triglycerides
PCSK9 Inhibitors	↓ LDL
Niacin	↓ LDL ↑ HDL



500 25

- 500 25



500 25

- 500 25

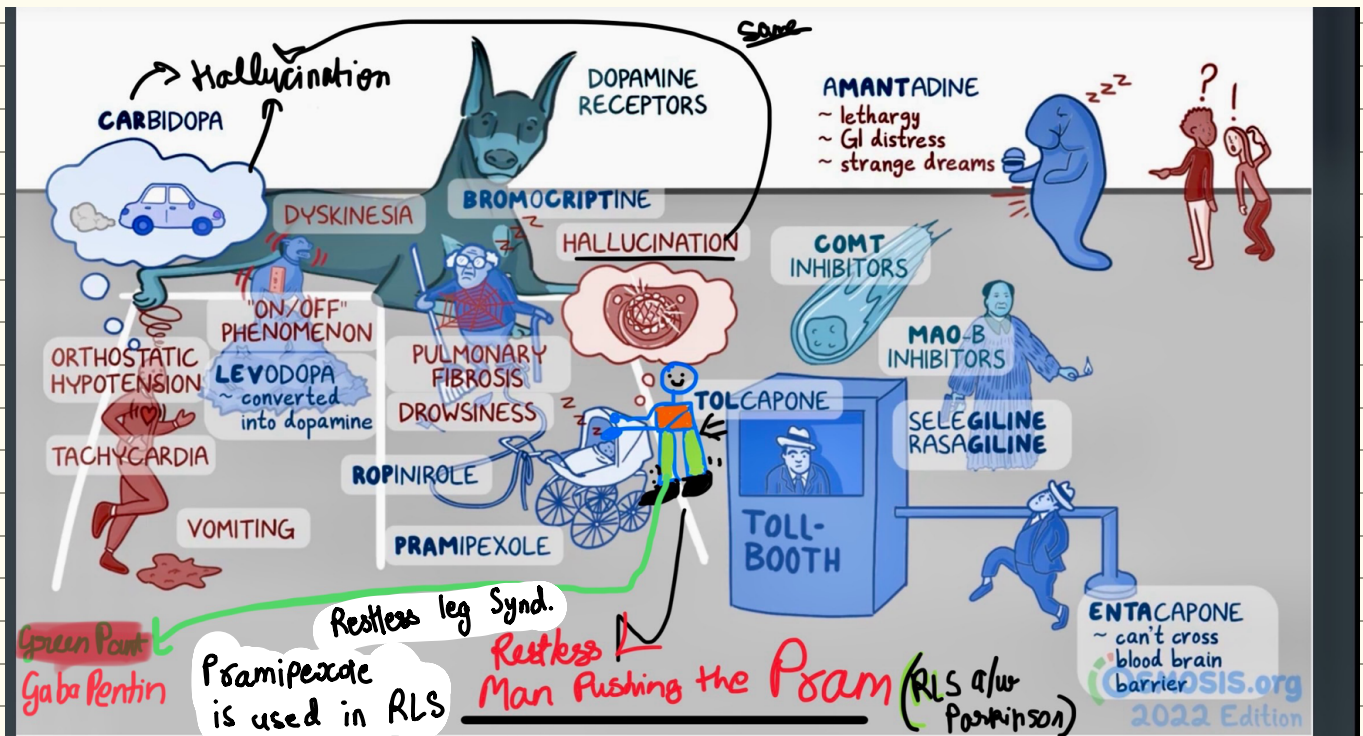
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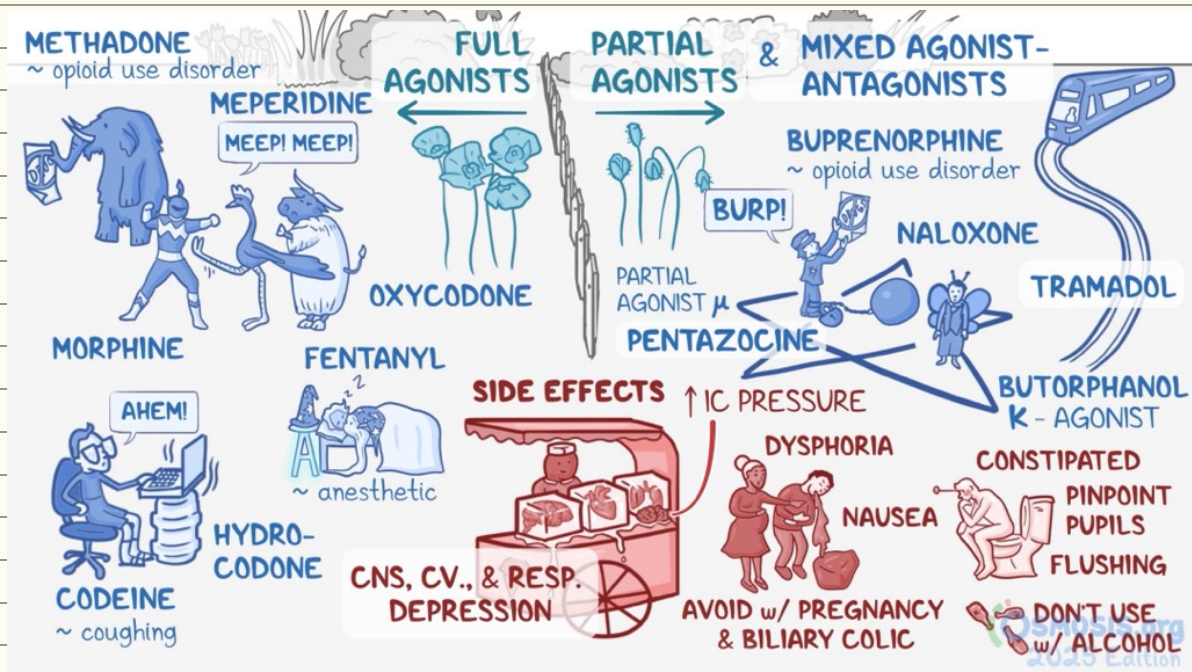
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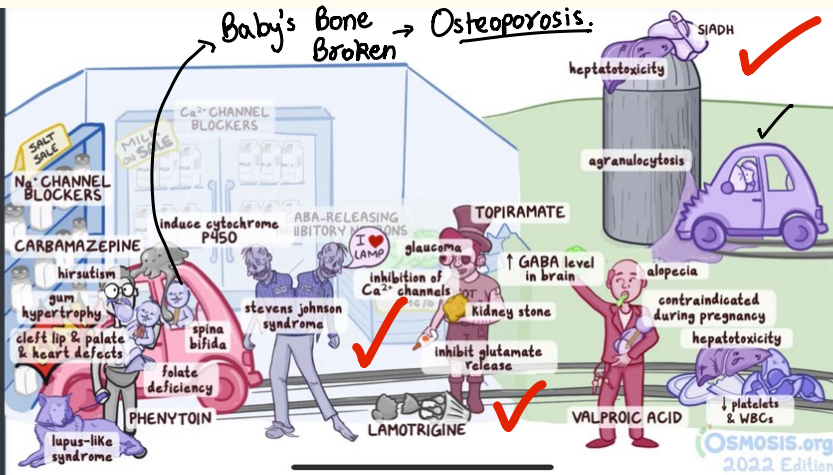
Anti-Parkinson Drugs.



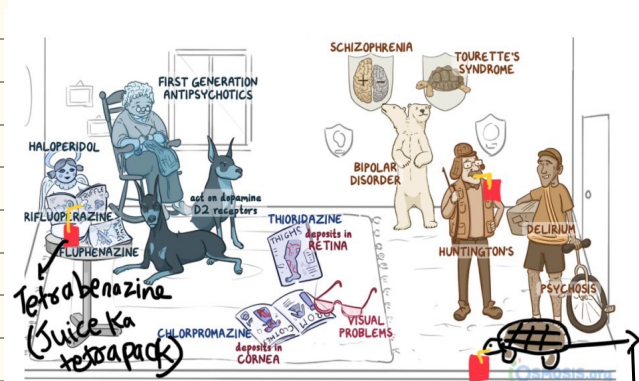
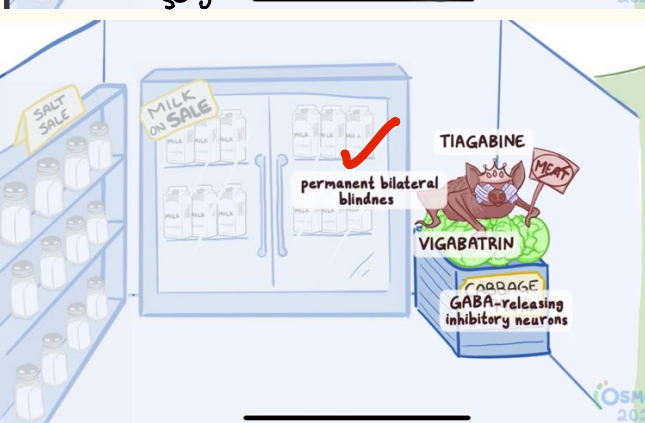
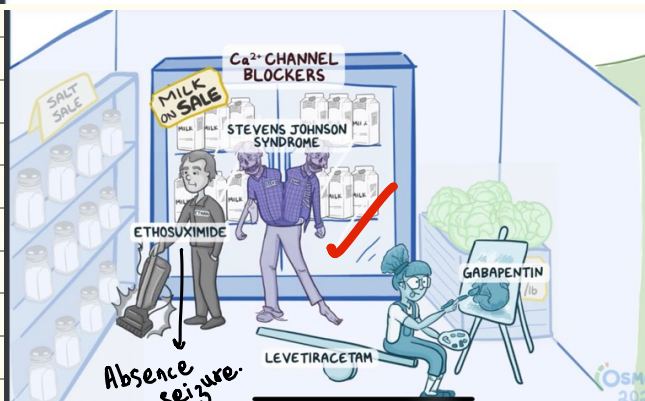
OPIOIDS - Heroin is an opioid.



Baby's Bone Broken → Osteoporosis.

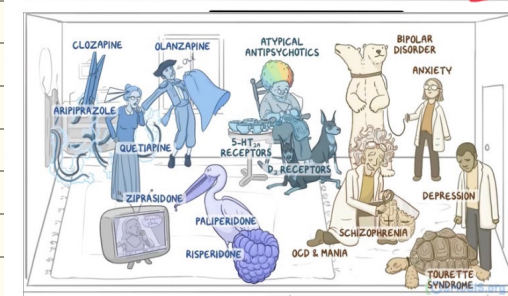


(Non BZO) ✓
 (Anti-convulsants)



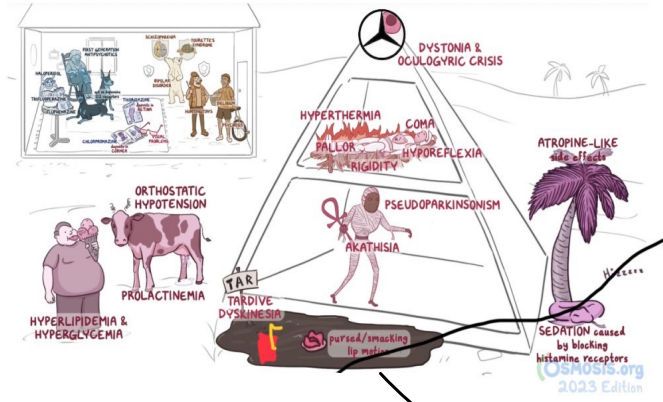
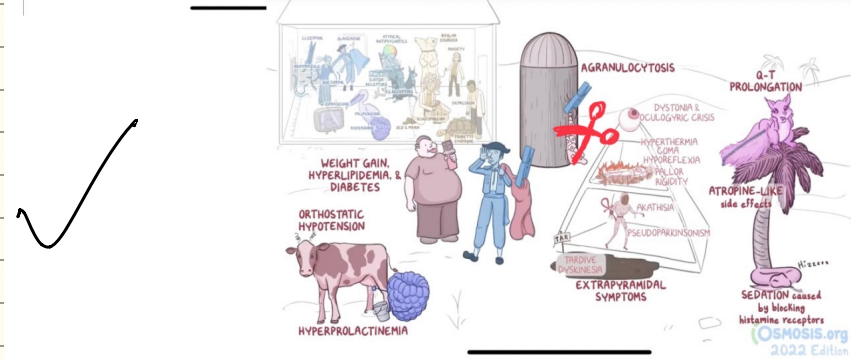
The Juice tetrapack

Jetrabenazine

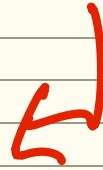


This tortoise is Tourette's Syndrome

& tetrabenazine is the t/c



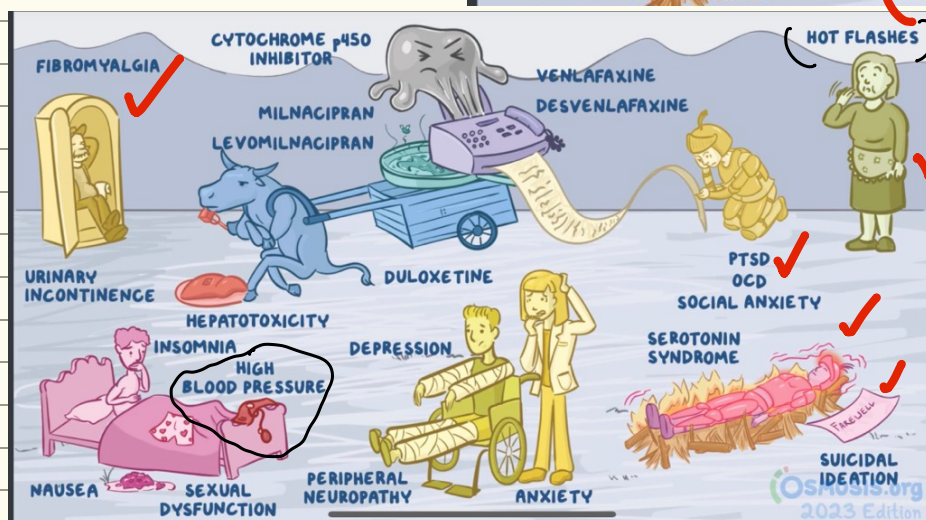
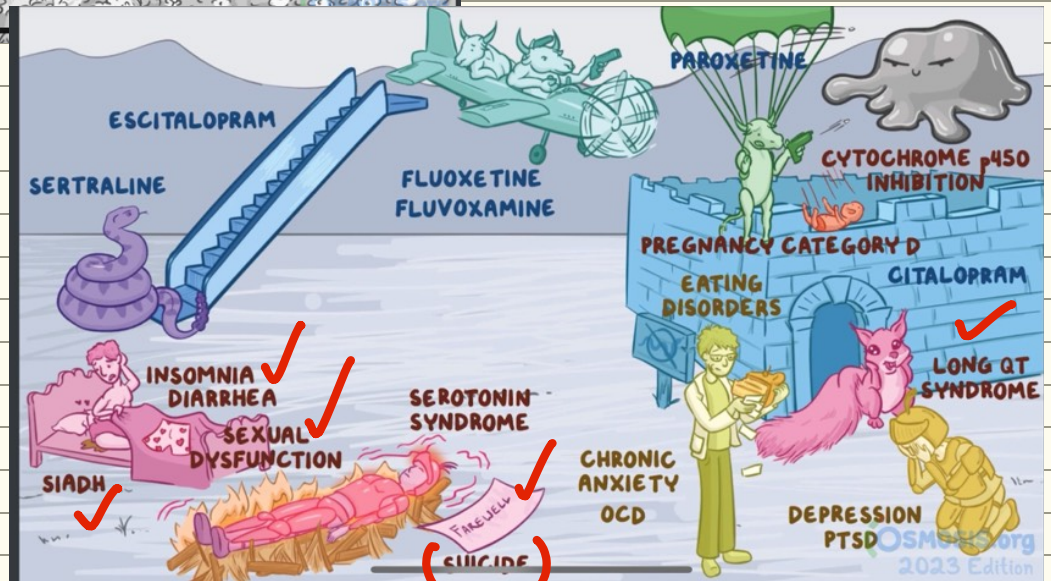
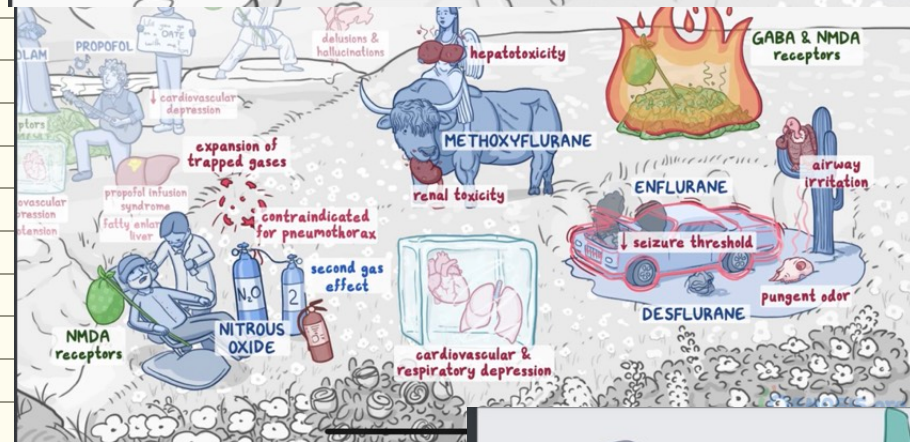
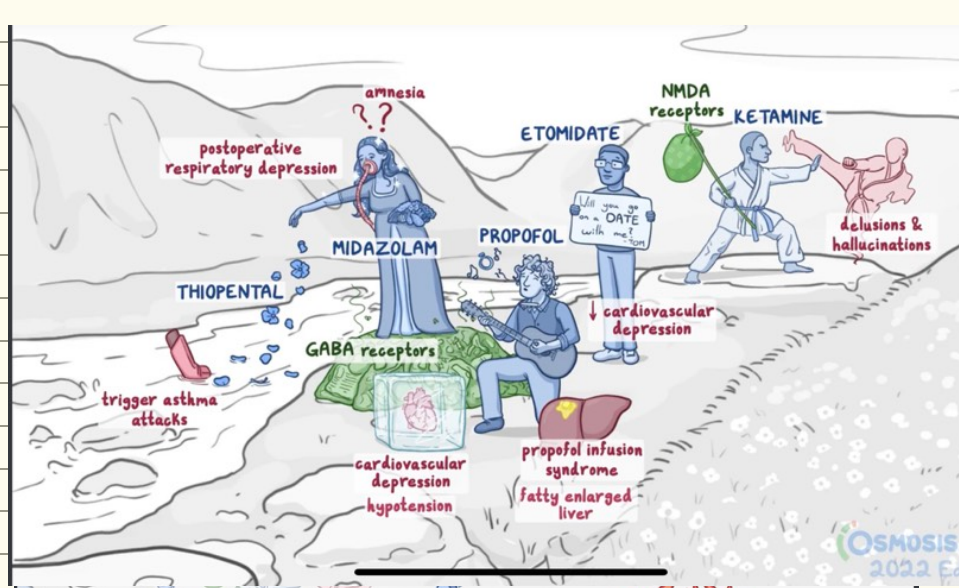
General Anesthetic



SSRI



SNRI

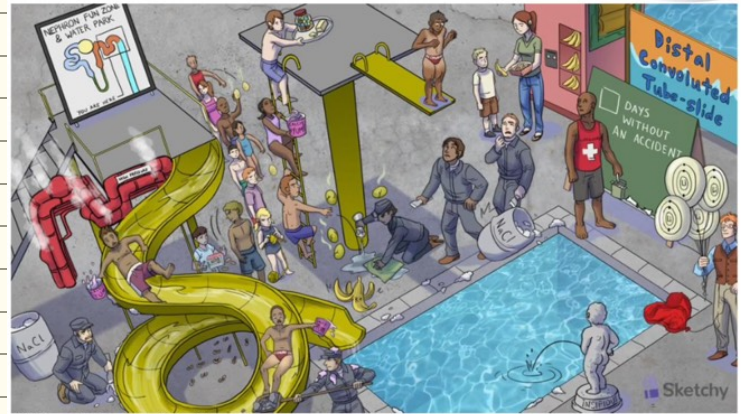




Loop Diuretics

1. Loop de Loop of Henle: Loop of Henle
2. Thick ascending Limb of the loop of Henle: site of action of most loop diuretics. Most relevant is sodium chloride blocks
3. Banana vending machine: Na^+/K^+ ATPase on the basolateral membrane, that will pump sodium into the interstitium
4. Three P batteries: ATPase
5. Yellow track: lumen of the renal tubule
6. Platform: intracellular compartment
7. Background wall is the interstitium
8. Track worker taking peanuts, bananas, and 2 chloride packets: $\text{Na}^+/\text{K}^+/\text{Cl}^-$ cotransporter (NKCC) reabsorbs these ions at the luminal membrane of the TAL thick ascending limb
9. Water secured in car: the TAL is impermeable to water (diluting segment)
10. Furious kid: furosemide (loop diuretics)
11. Furious kid clinging to food: furosemide selectively blocks the NKCC transporter on the luminal membrane of the TAL, keeping sodium in the lumen in the tubule representing furosemides ability to reduce reabsorption of NaCl causing natriuresis.
12. Loop diuretics are the most efficacious currently
13. Ethics: ethacrynic acid (loop diuretic)
14. Furious kid clinging to magnets and calci-yum ice cream: by blocking the NKCC, Loop diuretics reduce the lumen positive potential, promoting the excretion of Mg^{2+} and Ca^{2+}
15. Falling magnets: prolonged use of loop diuretics can cause hypomagnesemia, especially in diet deficient patients
16. Falling calci-yum ice cream: Loop diuretics can cause hypocalcemia (rare)
17. Pro-sluggers: prostaglandins
18. Furious kid welding pro-sluggers: loop diuretics induce the expression of COX-2, synthesizing prostaglandins that enhance salt excretion and dilate the afferent arteriole
19. Kid opening a path to afferent line of coaster: Prostaglandins will increase RBF in the afferent arteriole of the glomerulus. Thus enhancing diuretic action
20. Fire extinguisher inhibiting the pro-sluggers: NSAIDs decrease prostaglandin synthesis, interfering with the actions of loop diuretics
21. Failing heart balloon: loop diuretics are 1st line for the symptomatic treatment of acute decompensated heart failure with fluid overload, reduction for peripheral or pulmonary edema
22. Wet lungs: Loop diuretics cause maximal amount of diuresis in the shortest amount of time. Used 1st line in acute heart failure with orthopnea that has crackles in the lungs and JVD. Loops will help with PRELOAD function. This will not help prolong the life of the heart.
23. Yellow inner tube: loop diuretics treat ascites in liver failure
24. High pressure pipes: loop diuretics can be useful in the treatment of HTN
25. Diuretics lower blood pressure by decreasing body sodium stores
26. Banana peel: loop diuretics are potassium wasting causing hypokalemia, hypokalemia can exacerbate any underlying arrhythmias in heart failure
27. Loud gong: loop diuretics can cause dose related hearing loss
28. Stinky sulfur eggs: most loop diuretics are sulfur drugs
29. Sulf-a-less ethics: Ethacrynic acid is not a sulfu drug
30. Kidney filled with blue tickets: Interstitial nephritis can be caused by loop diuretics
31. Knitting needles: loop diuretics can cause hyperuricemia, may lead to gouty arthritis
32. Park employee cleaning the floor with contracted bleach bottle: loop diuretics can cause contraction alkalosis, causing dehydration and metabolic alkalosis by many mechanisms.

LOOP



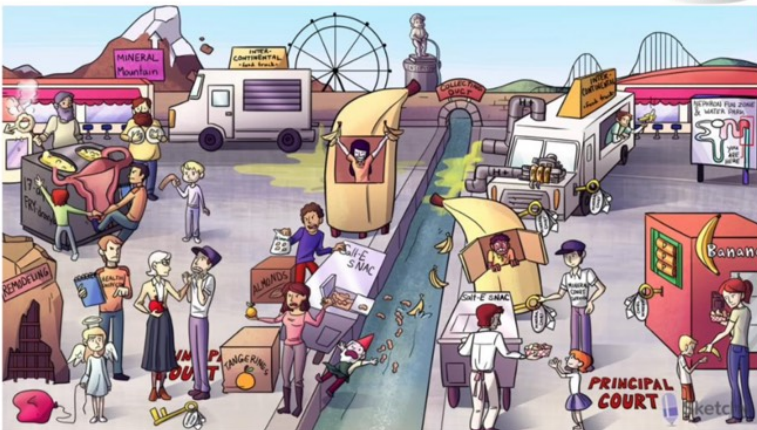
Thiazides - Distal Convoluted Tube-Slide

1. Distal convoluted tube slide: Distal convoluted tubule
2. Distal convoluted tubule: site of action for thiazide diuretics
3. Banana vending machine: Na^+/K^+ ATPase on the basolateral membrane
4. Three P batteries: ATPase
5. Yellow tube slide: tubular lumen
6. Area outside slide: intracellular compartment
7. Sodium chloride salt scraper: NaCl cotransporter reabsorbs these ions at the apical membrane of the DCT
8. Active slider dropping the calci-yum ice cream: calcium is actively reabsorbed at the DCT (regulated by PTH)
9. Chloro-thighs, thiodore Roosevelt on high dive: Hydrochlorothiazide and chlorthalidone (thiazide diuretics)
10. Sodium chloride dumping into pool: thiazides inhibit NaCl reabsorption by blocking the NaCl cotransporter on the apical membrane (causing natriuresis)
11. Chloro-thighs kid dropping calci-yum: thiazide diuretic enhance calcium reabsorption, may be result at proximal and distal tubule. At the proximal tubule thiazide induced volume depletion leads to enhanced sodium and passive calcium reabsorption. At the distal tubule thiazides block sodium entry into the epithelial cell. This decrease of sodium entry enhances sodium calcium exchange at the basolateral membrane leading to the enhanced absorption of calcium
12. High pressure pipes: thiazide diuretics are one of the first line treatments for mild or moderate HTN
13. Loop diuretics are first line for acute, not thiazide
14. Floppy failing heart balloon: use of thiazides can be useful in the symptomatic treatment of heart failure (loop diuretics are first line)
15. Inispidus fountain: thiazide diuretics treat nephrogenic diabetes insipidus, thiazide diuretics can reduce polyuria and polydipsia in nephrogenic DI, this paradoxical association is due to a hypovolemia induced increase in Na and H_2O reabsorption in more proximal segments of the nephron where
- ADH is not working in the distal tubule. Thus allowing for more electrolytes to be retained.
16. Removing tube slide stones: thiazide diuretics can be used to prevent calcium stones (increased calcium reabsorption causes hypocalcemia) found in hyperparathyroidism, sarcoidosis, New calcium chalk: thiazide diuretics may benefit patients with osteoporosis
17. Elevated calci-yum ice cream: thiazide diuretics can cause hypercalcemia
18. Elevated Candy jar and stick of butter: Thiazide diuretics can promote Hyperglycemia and also hyperlipidemia.
19. HIGHdrolchlorothiazide and the HIGH dive raises a lot of lab values
20. Yellow knitting needles: thiazide diuretic can cause hyperuricemia (can precipitate gout) due to hypovolemia and hyper absorption of uric acid
21. "Lift"ium balloons: thiazide diuretics decrease the amount of lithium cleared, therefore there will be increased serum lithium levels
22. Grey kid pissing in the pool: Lithium is a common cause of nephrogenic diabetes insipidus
23. Potassium depleted banana peel: Thiazide diuretics block the Na^+/Cl^- cotransporter in the distal convoluted tubule, increasing sodium delivery to the collecting duct. This leads to increased potassium secretion by the collecting duct in exchange for Na^+ reabsorption leading to hypokalemia
24. Spilled peanut shells: thiazides diuretics can cause hyponatremia
25. Rotten eggs being tossed: Thiazides are sulfur drugs
26. Contracted bleach bottle: thiazides can cause contraction alkalosis, metabolic alkalosis by hypovolemia induced renin production by the kidneys. Increasing aldosterone which causes increased H^+ excretion at the collecting duct and more ATII leading to increased sodium bicarb reabsorption at the proximal convoluted tubule

Will increase aldosterone

Can lead to gout

THIAZIDES



K⁺ sparing Diuretics - Pt 1

1. Central gutter: work in the collecting duct
2. Collecting duct (site of action of the K⁺ sparing diuretics)
3. Mineral-O-Food court: mineralocorticoids site of action (aldosterone) in the collecting duct
4. Principal court: principle cell of the collecting duct (major site of $\text{Na}^+/\text{K}^+/\text{H}_2\text{O}$ transport)
5. Banana vending machine: Na^+/K^+ ATPases on the basolateral membrane, help pump reabsorbed sodium into interstitium
6. Three P batteries: ATPase
7. Food court ground: intracellular compartment
8. Salt-E sNaC cart: epithelial Na^+ channels (ENaC) reabsorb Na^+ across the luminal membrane of the collecting duct and distribute to the interstitium
9. Water in gutter: tubular lumen
10. Banana stand dumping bananas: K⁺ channels allow the excretion of K⁺ across the luminal membrane of the collecting duct
11. Salt-E sNaC cart: epithelial Na^+ channels (ENaC) reabsorb Na^+ creates a negative luminal potential that facilitates K⁺ excretion, this is due to the negative electrical potential caused by the absorption of sodium into the interstitium that will draw the potassium into the tubular lumen
12. Alpha intercontinental food truck: alpha intercalated cell of the collecting duct (major site of H^+ excretion)
13. Batter powered acid pump: H⁺ATPase on the apical membrane of the alpha intercalated pumps H^+ into the lumen
14. Three P batteries: ATPase
15. Mineral court services: intracellular mineralocorticoids (aldosterone) receptor
16. Mineral key: aldosterone binds to the salt-E sNaC upregulating ENaCs on the apical membrane, increasing Na^+ reabsorption
17. Mineral Key activating the banana vending machine: aldosterone upregulates Na^+/K^+ ATPase on the basolateral membrane
18. Mineral key activating the banana stand: aldosterone upregulates K⁺ channels on the apical membrane, increasing K⁺ excretion
19. Loops and thiazides activate renin (and aldosterone) due to hypovolemia and cause a hypokalemia because they leave all of the sodium in the tubular lumen until it gets to the collecting duct. Then there is an attempt by the ENaC to retain all of the sodium at the expense of potassium, and facilitate H⁺ secretion causing a metabolic alkalosis.
20. Tangerines: triamterene (K⁺ sparing diuretic)
21. Tangerines blocking the salt-E sNaC cart: triamterene inhibits Na^+ reabsorption through ENaC
22. Almonds: amiloride (K⁺ sparing diuretic)
23. Almonds blocking the Salt-E sNaC cart: amiloride inhibits Na^+ reabsorption through ENaC
24. Salty sodium peanuts falling into the duct: K⁺ sparing diuretics inhibit Na^+ reabsorption at the collecting duct, promoting natriuresis
25. Apple with the teacher: eplerenone (K⁺ sparing diuretic)
26. Teacher with apple antagonizing the mineral court services man: eplerenone antagonizes the mineralocorticoid receptor
27. Health inspector with Spiral bound notebook: spironolactone (a K⁺ sparing diuretic)
28. Health inspector antagonizing the mineral court services man: spironolactone antagonizes the mineral corticoid receptor
29. Crumbling mineral mountain: K⁺ diuretics (spironolactone, eplerenone) are useful in the treatment of 1 and 2 hyperaldosteronism, conn syndrome, ACTH etc...
30. Failing heart balloon: K⁺ diuretics (spironolactone, eplerenone) are useful in the treatment of heart failure to prevent K⁺ wasting
31. Remodeling: mineralocorticoid antagonists (spironolactone, eplerenone) prevent myocardial remodeling induced by high level of aldosterone
32. Angel: mineralocorticoid antagonists decrease mortality in heart failure
33. Inispidus fountain: amiloride is useful in the treatment of Li⁺ induced nephrogenic diabetes insipidus, this will block lithium entry into collecting duct cells increasing clearance of lithium

17-fryer... can lead to gynecomastia

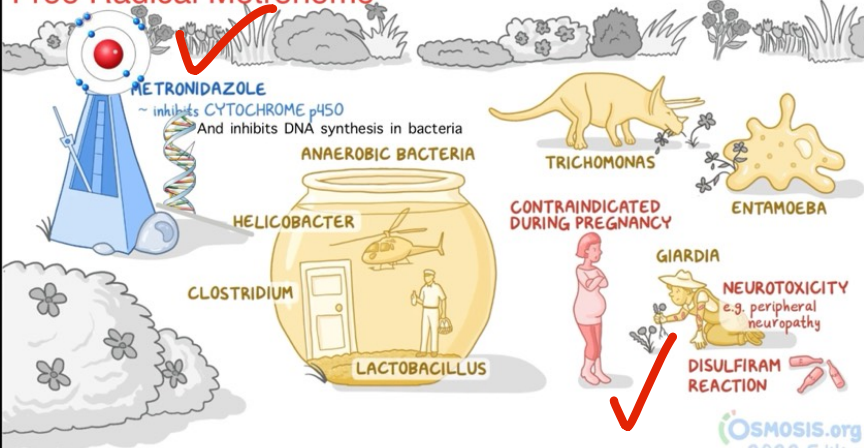


K⁺ Sparing Diuretics - CONT

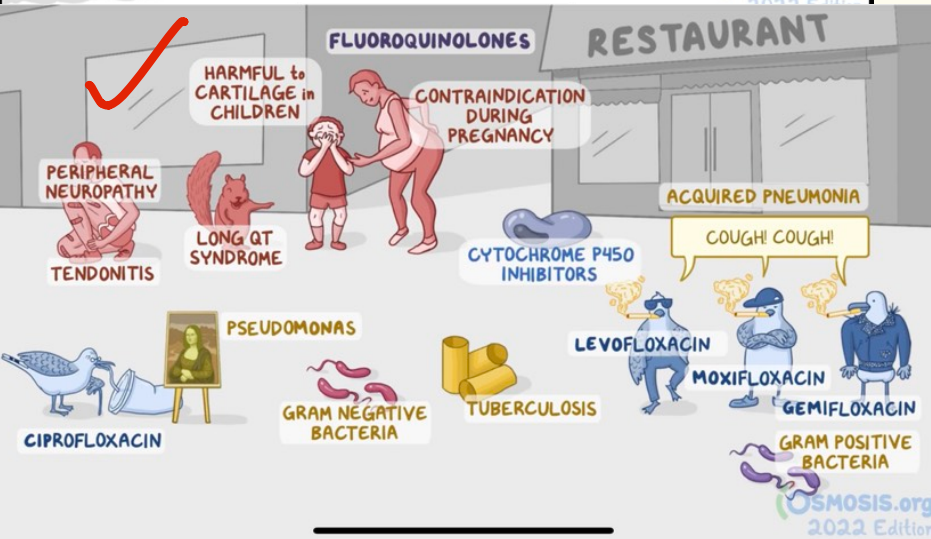
34. Little gnome blocked by almonds and tangerines: amiloride and triamterene are useful in the treatment of Liddle's syndrome (overactive ENaCs)
35. Elevated banana's: K⁺ sparing diuretics can cause mild or even dangerous hyperkalemia, seen in renal disease when there is a decreased excretion of potassium and with drugs that decrease renin and angiotensin activity such as Beta blockers and ACE inhibitors. Use a potassium wasting diuretic with these pts
36. Acid spill: K⁺ sparing diuretics cause a normal anion gap metabolic acidosis (by decreasing the function of the H⁺ATPase) by inhibition of aldosterone's effects at the collecting duct
37. Worker holding 4 acid tubes: K⁺ sparing diuretics inhibit the effects of aldosterone in the collecting duct causing a type 4 renal tubular acidosis
38. Big K: type 4 RTA is associated with hyperkalemia: this is the only one associated with hyperkalemia.
39. Spironolactone is much less selective with the aldosterone receptor, spironolactone is a synthetic steroid that can bind to other steroid receptors and processing enzymes. This has anti androgenic side effects and can block testosterone synthesis.
40. Fried male symbol: testosterone produced from cholesterol
41. Health inspector inhibiting 17 alpha "fry" droxylase: spironolactone inhibits 17alpha-hydroxylase. Important in the adrenal cortex and testes
42. Bubbling ovary shaped vats: spironolactone treats the symptoms of androgen excess in polycystic ovarian syndrome, used after a trial of birth control
43. Bushy beard: symptoms of androgen excess (hirsutism) in PCOS are treated with spironolactone
44. Preventing boy from receiving onion ring: spironolactone directly antagonizes the androgen receptor
45. Lids on chest: Gynecomastia caused by spironolactone
46. Droopy churro: Spironolactone can cause impotence and decreased libido

Spironolactone

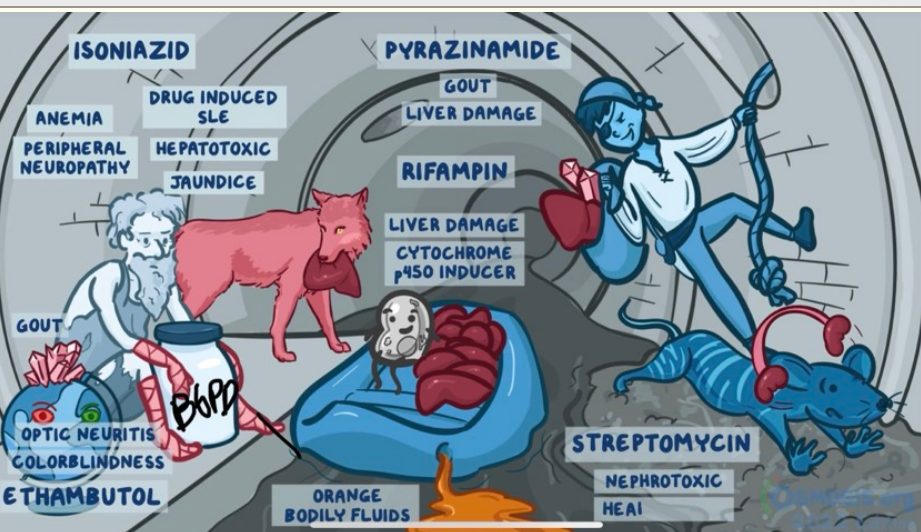
Free Radical Metronome



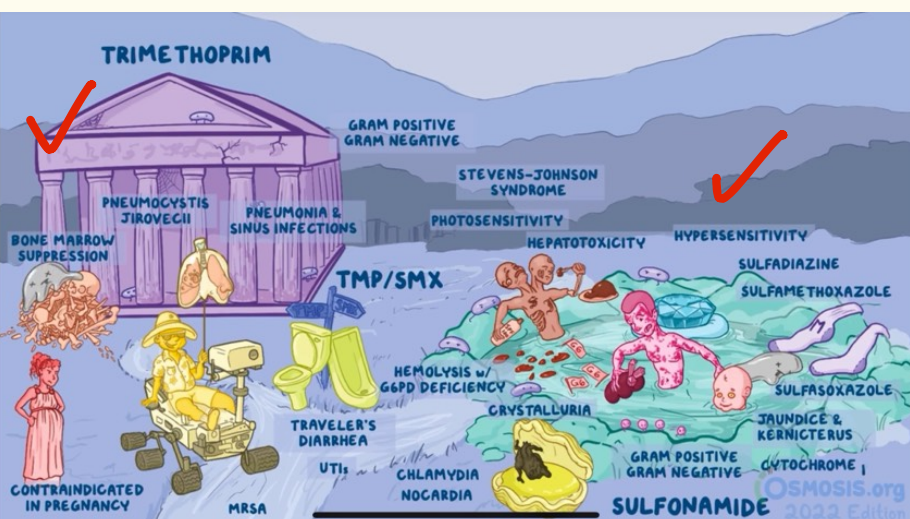
Metronidazole



FLUOROQUINOLONE



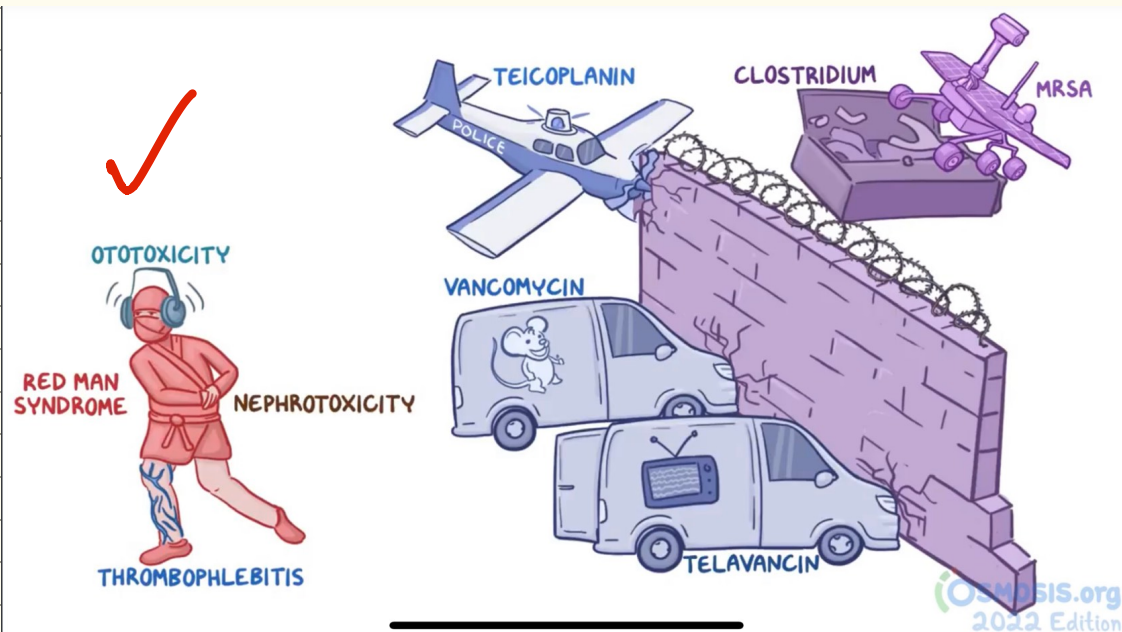
Anti-TB drugs.



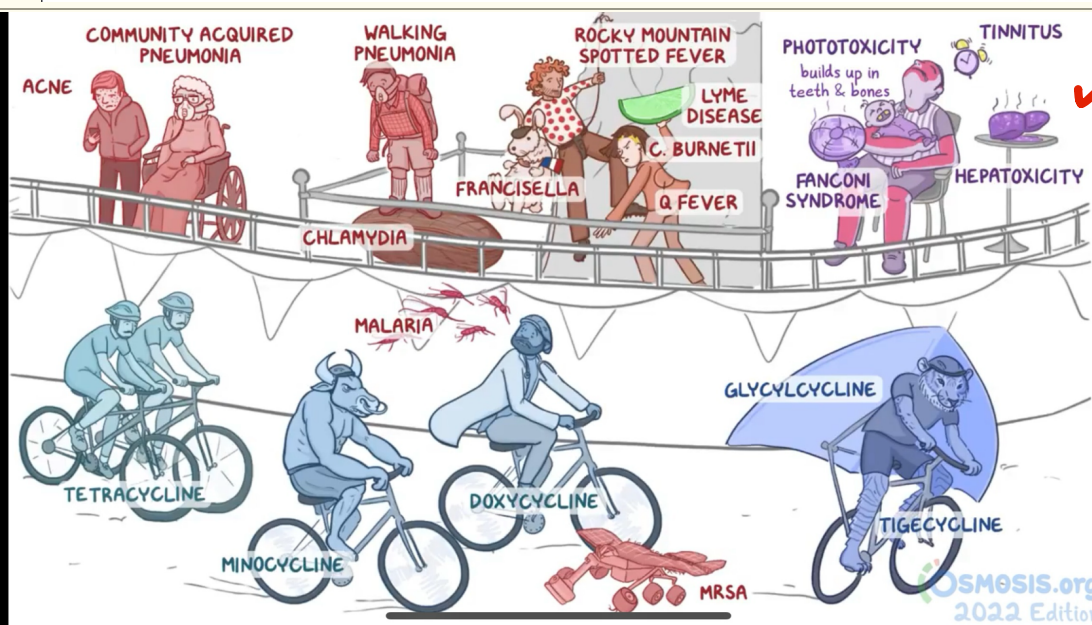
TMP-SMX



Anti-Histamine



Vancomycin.



Tetracycline

